Disorders of Potassium

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KEYWORDS

• Potassium • Hyperkalemia • Hypokalemia • ECG changes

KEY POINTS

- Potassium concentration is tightly regulated in the body and disruption of potassium-related ion gradients increase morbidity and mortality.
- Hyperkalemia and hypokalemia can both lead to life threatening arrhythmia and should be treated quickly and efficiently.
- Treatment for hyperkalemia involves a multimodal approach: attempting to stabilize the cardiac membrane, shift potassium into cells, and ultimately remove excess potassium from the body.
- Treatment for hypokalemia involves replacing lost potassium and preventing further losses, often requiring medication adjustment and identification of the cause of the potassium deficit.

INTRODUCTION

Potassium derangements are commonly encountered in the emergency department (ED) ranging from mild presentations with vague symptoms to severe cases that are life-threatening. As many as one in eleven patients presenting to the ED will have either hypokalemia or hyperkalemia.[1] Many derangements in potassium are not a result of a primary electrolyte disorder, but rather a sequela of an underlying disease process or drug interaction. In the ED, the key to managing these patients will be to identify life-threatening derangements of potassium, provide emergency stabilization, and coordinate definitive treatment.

BACKGROUND

Maintenance of strict serum potassium levels is critical to multiple cellular functions and potassium is tightly regulated, with 98% stored in the intracellular space.[2] Multiple factors are involved in homeostasis. In most patients, the kidneys account for 90% of

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potassium elimination and the remaining 10% is eliminated through the gastrointestinal tract. In patients with end-stage renal disease (ESRD), gut elimination is increased by 250% to account for altered renal physiology.[3] Rarely, dietary potassium supplements may cause a derangement in serum levels. Required daily intake of 2000 to 3500 mg (equivalent to 50–90 mEqs) is unlikely to cause issues in patients with normally functioning kidneys.[4] Though unlikely, dietary intake of potassium supplements or foods high in potassium may cause dangerous or deadly elevations in serum potassium levels especially in patients with underlying renal disorders. Common culprits are non-sodium-based seasoning, bananas, potatoes, avocados and beans.[5]

Renal handling of potassium is primarily governed by aldosterone at the distal nephron.[6] The primary action of aldosterone is to increase blood pressure through sodium absorption and potassium elimination. Aldosterone's influence is mediated via renal blood flow, sodium delivery, sympathetic tone, renin-angiotensin-aldosterone system (RAAS) activity, serum potassium concentration, acid-base status, and a myriad of drugs (**Table 1**).[7] Additional potassium regulation occurs via transcellular shifts and non-aldosterone-mediated renal mechanisms. Potassium disorders can occur from a disruption of regulatory physiology due to renal injury and/or drug interactions and, rarely, genetic channelopathies.[8] Occasionally, multiple drug interactions may exacerbate underlying renal disorders resulting in significant and deadly derangements of potassium. For example, the antibiotic trimethoprim-sulfamethoxazole (TMP/SMX), commonly used to treat urinary tract infections, carries a risk of hyperkalemia in 51 per 100,000 patients. However, this risk increases sevenfold when prescribed to an elderly patient on an angiotensin-converting enzyme inhibitor (ACE-I) or angiotensin receptor blocker (ARBs).[9,10]

ASSESSMENT

In addition to a detailed history and physical, a patient suspected of potassium derangement should receive both an electrocardiogram (ECG) as well as the measurement of serum electrolytes. The electrocardiogram is inexpensive, instantaneous, easily repeatable, and cues the provider into the cardiac manifestation of hypo- or hyperkalemia. The large gradient of intracellular potassium concentration to extracellular potassium helps maintain a tightly regulated negative resting membrane potential, working to stabilize myocytes and prevent spontaneous action potentials. Abnormalities in serum potassium concentration affect cardiac myocyte excitability

Table 1
RAAS altering medications

Drugs Associated with Hyperkalemia		
RAAS Altering	**Drugs**	**Example**
	Adrenergic Blockers	Metoprolol
	Direct Renin Blockers	Aliskren
	NSAIDS	Ibuprofen
	ACE Inhibitors	Lisinopril
	ARBs	Valsartan
	Aldosterone Blocker	Spironolactone
	Heparin Analogs	Heparin
	Calcineurin Inhibitors	Cyclosporin
Renal Elimination	Potassium Sparing Diuretics	Amiloride
	Antibiotics	Trimethoprim
Cellular Shift	Cardiac Glycosides	Digoxin
	Depolarizing Paralytics	Succinylcholine

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in several ways, resulting in potentially characteristic ECG changes or cardiac arrhythmia.[11] These abnormalities may prompt emergency stabilization while laboratory values are being obtained.

HYPERKALEMIA

Defintion/burden of disease

In most laboratory assays, hyperkalemia is defined by a serum value greater than 5.0 mmol/L.[12] Severe hyperkalemia is defined by a serum value of 6.0 mmol/L with ECG findings, or an absolute value of 6.5 mmol/L or more regardless of ECG findings. Hyperkalemia that causes ECG change puts the patient at least at a moderate risk of poor outcome.[13] An evaluation of patients admitted to the ICU with hyperkalemia showed that mortality was higher in those with normal baseline renal function. This is indicative of patients with CKD developing tolerance to higher potassium levels over time, and acute hyperkalemia with ECG changes, regardless of the absolute serum value, can be more deadly than chronic hyperkalemia with no cardiac manifestation. Mortality in patients with hyperkalemia in this study was also associated with concurrent infections, multisystem organ failure, and bleeding disorders.[14]

Evaluation

Potassium disorders are diagnosed by laboratory assessment; therefore, one must ensure the reported value is correct. Pseudo-hyperkalemia is a condition where laboratory analysis reveals a falsely elevated potassium due to improper handling or acquisition of the sample.[15] Though many common laboratory assays can detect hemolysis, one must interpret the value reported in the correct clinical context. Common reasons for hemolysis include: stationary time before laboratory analysis, sheer force during blood acquisition through the venipuncture kit, or prolonged torniquet time.[16] Certain blood cancers can cause pseudo-hyperkalemia due to the fragility of the immature cancer cells and may not trigger laboratory hemolysis warnings.[17] To quickly assess if this hyperkalemia is true, consider your pretest probability and have a low threshold to repeat the test if suspicious for lab error. Additional information may helpful when trying to gauge if the laboratory result is spurious, such as the patient's ECG, if there are other laboratory results that are physiologically unlikely, or extreme potassium elevation with reported normal renal function. Another way to quickly evaluate the potassium level would be a rapid point-of-care (POC) test, if available. Unfortunately, some of these POC tests may be inaccurate compared to serum potassium levels, with values differing by a mean of 0.5 mmol/L.[18] It is reasonable to utilize POC tests in conjunction with confirmatory serum values and an ECG to risk stratify of pseudo-hyperkalemia versus true hyperkalemia. While waiting to confirm if pseudo-hyperkalemia is present, it is reasonable to assume the patient has true hyperkalemia and start treatment with cardiac stabilization while waiting on confirmatory testing.

Electrocardiogram

Traditionally, it was thought that the ECG changes related to hyperkalemia evolve in a sequential manner and serum levels can be predicted by the ECG changes (**Fig. 1** and **Table 2**).[19] This was found to be untrue, however, ECG manifestations do offer clues as to the body's response to the electrolyte change and how vulnerable the myocardium may be to arrythmias. The ECG alone is neither sensitive nor specific enough to make the diagnosis or reflect the severity of hyperkalemia. Reliance on the ECG alone may miss up to 50% of cases of hyperkalemia.[20] Any of the classic ECG findings may

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Fig. 1. ECG changes in hyperkalemia. (*Courtesy of* Richard Jean-Louis, MD. K+ = 9.2)

occur at any potassium level, but QRS widening, bradycardia and ventricular-based rhythms are associated with an increased risk of adverse events including mortality.[21] A retrospective analysis of admitted patients with hyperkaemia showed that patients who died were more likely to have traditional ECG manifestations of hyperkalemia than those who survived.[14]

Fig. 2 shows a patient with a history of ESRD who presented for weakness. The patient's potassium was 9.2 and the ECG showed classic manifestation of potassium's cardiac toxicity with an extreme right axis deviation, wide QRS, loss of P wave, and peaked T waves. It is important to not confuse this ECG with ventricular tachycardia as antiarrhythmic administration can be fatal. The patient was started on cardiac membrane stabilization, potassium lowering therapy, and emergent nephrology consult. The subsequent ECG (shown in Fig. 3) reflects a potassium of 7.0.

MANAGEMENT

The treatment of hyperkalemia depends not only on the absolute laboratory value but also the clinical manifestations of hyperkalemia. Treatment is generally divided into phases: emergency stabilization, transcellular shifting, and elimination. The treatment of hyperkalemia is as important as the evaluation of the underlying cause (Table 3).

Table 2
ECG changes in Hyperkalemia[20]

Typical ECG Manifestations	Severity
Peaked T Waves	Mild
PR Changes	Moderate
QRS widening Loss of P wave	Severe
Sine Wave morphology Fatal rhythm	Critical

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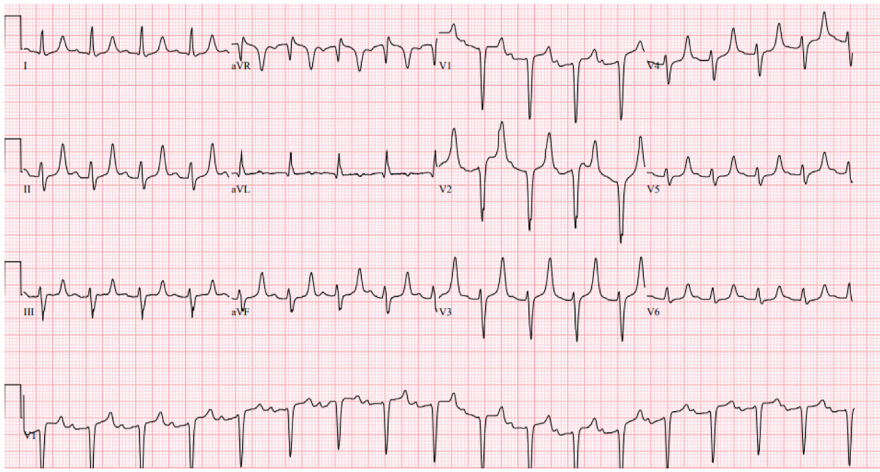

Fig. 2. ECG changes in hyperkalemia. (*Courtesy of* Richard Jean-Louis, MD. K+ = 7.0)

Emergency stabilization

The emergent stabilization for hyperkalemia involves the restoration of the cardiac resting membrane potential. When the extracellular concentration of potassium rises it causes membrane depolarization. In cardiac cells, this spontaneous depolarization leads to changes that manifest itself on the ECG which should guide stabilization therapy.

The first step of stabilization involves the administration of calcium if there are any ECG changes, especially those associated with poor outcomes. Intravenous calcium comes in two forms: calcium chloride and calcium gluconate. One gram of calcium chloride contains three times the amount of elemental calcium compared to 1 g of calcium gluconate. Initially it was thought that calcium gluconate required hepatic metabolization to release elemental calcium, but a study of intraoperative liver transplant patients with liver transplants showed equal rises in calcium levels even without any

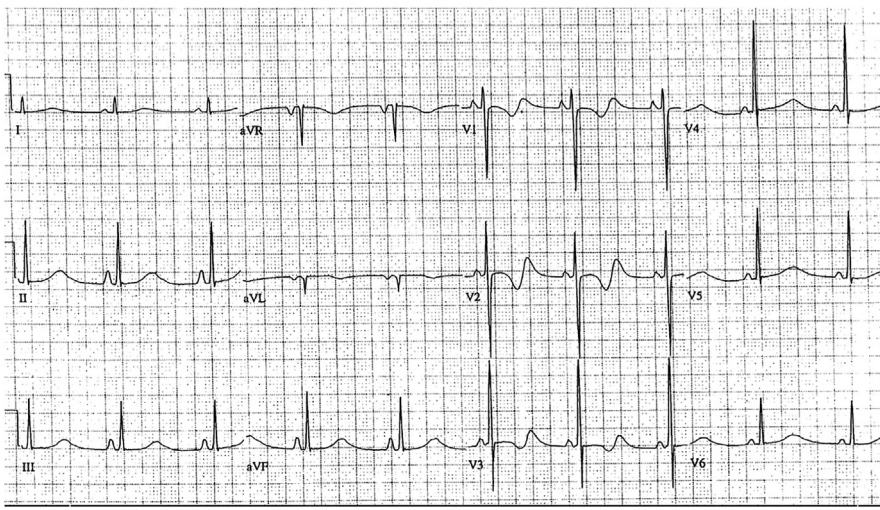

Fig. 3. Nikelekam T-waves. Inverted T-waves followed by a large U-wave. (*Courtesy of* Amal Mattu, MD)

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Table 3
Treatment of hyperkalemia

Medication	Dosing	Onset	Duration	Mechanism
Calcium Gluconate	2–3 g	1 min	30–60 min	Membrane Stabilization
Calcium Chloride	1–2 g	1 min	30–60 min	Membrane Stabilization
Insulin	5–10 units IV	10 min	2–6 h	Intracellular shift
Albuterol	10–20 mg nebulized	3 min	1–4 h	Intracellular shift
Sodium Bicarbonate	variable	30 min	2–6 h	Intracellular shift
Furosemide	variable	5 min	2–6 h	Kaliuresis
Patiromer	8.4 g (initial)	7 h	Up to 48 h[a]	GI elimination
Sodium zirconium cyclosilicate	10 g (initial)	1 h	Up to 48 h[a]	GI elimination

[a] may require repeat dosing.

liver function.[22] Historically, calcium chloride has been avoided except in the peri-arrest period or in patients with central access due to its risk of tissue necrosis in extravasation. However, the administration of calcium chloride in reliable, large-bore peripheral access has been shown to be safe in observational studies[23] If calcium is given it should be given in an amount needed to change the ECG interval that prompted its administration (QRS widening, ventricular rhythm and so forth). Traditional dosing is 1 to 2 g of calcium chloride, but if calcium gluconate is given then at least 2 to 3 g may be needed. The onset of action should be within 5 minutes and last for 30 to 60 minutes. Repeat dosing may need to occur if the serum potassium level has not been lowered.[24] The emphasis should be on safe administration, appropriate dosing, and evaluation for effect.

If there are no cardiac manifestations of hyperkalemia on ECG and the absolute value is not at a severe range, then withholding calcium therapy may be reasonable. Potential downsides to calcium administration are mainly extravasation injuries and long-term risk of calciphylaxis, although single time dosing for an acute condition does not clearly lead to this.[25]

TRANSCELLULAR SHIFT

The next phase in the management of hyperkalemia involves intracellular shifting of potassium. The goal of the therapy is to temporarily shift the potassium into the cell, therefore reducing the effects of elevated serum potassium until it can be eliminated later. The mainstay of therapy here involves the manipulation of the Na-K-ATPase pump moving potassium into the cell.

Intravenous (IV) insulin is often the first recommended treatment to shift potassium intracellularly. Insulin is often given along with glucose containing solution to prevent hypoglycemia, though giving additional glucose can be deferred in the setting of severe hyperglycemia. Consideration of the patient's fluid status is needed when choosing the concentration of glucose to administer with the insulin. In patients who are prone to have poor handling of excess volume (heart failure, ESRD, pulmonary HTN), consider giving the lower volume 50% dextrose ampule (D50; 25g dextrose per 50 mL ampule) rather than 10% dextrose in a larger volume to achieve the same amount of dextrose given (25g dextrose in 250 mL D10 bolus). This decision needs to be weighed against the risk of extravasation through poor peripheral access

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that is well documented with D50 administration. Fingerstick glucose levels need to be followed for at least 6 hours to monitor for hypoglycemia.

Insulin administration in the setting of renal dysfunction poses a unique challenge as insulin is renally cleared. In a single center study, patients with severe renal dysfunction but not on dialysis, the administration of 5 units versus 10 units of insulin IV showed similar rates of hypoglycemia but the larger dose was more effective in lowering potassium (0.9 vs 0.6 mmol/L).[26] Alternatively, other retrospective studies have shown increased rates of hypoglycemia utilizing the 10 units instead of 5 units.[27] Therefore in patients with hyperkalemia, starting with 5 units of insulin IV is reasonable to avoid hypoglycemia and allow for repeat dosing if needed.

Insulin administration exerts maximal effect when utilized in conjunction with beta-agonists, allowing for further activation of the Na-K ATPase pump.[28] Commonly, this involves the administration of nebulized albuterol. The dose to achieve appropriate transcellular shifting of potassium is almost 5 to 10 times the dose given for typical bronchodilation (10–20 mg for hyperkalemia vs 2.5 mg for asthma). The effect is seen within 90 to 120 minutes depending on the dose used.[29] Alternatives to inhaled beta agonism include terbutaline and epinephrine, which can be given by several routes. Epinephrine alone is unlikely to produce enough clinically significant shifting as stand-alone therapy but can be considered in the event of bradycardia or shock as hyperkalemia is a known myocardial depressant.[30] The negative effects of beta agonism mainly include tachyarrhythmia, anxiety, palpitations, and jittery sensation.

Sodium bicarbonate theoretically takes advantage of both potassium shifting as well as the renal elimination of potassium. However, multiple randomized controlled trials show little improvement with 8.4% bicarbonate ampule push administration unless, perphaps, administerd with insulin.[31] Alternatively, isotonic bicarbonate infusions (150 mL of 8.4% sodium bicarbonate in 1L of D5W) in the setting of metabolic acidosis may provide necessary potassium lowering therapy.[32] This may promote potassium dilution, potassium shifting, and renal potassium elimination. Combining bicarbonate with dextrose-containing fluids for a patient receiving insulin therapy can help provide another buffer to avoid hypoglycemia. This does require a 1 to 2 L volume administration which may not be an option in patients at risk for volume overload, though could be considered as part of the resuscitative strategy in a patient believed to be hypovolemic with metabolic acidosis and hyperkalemia.

ELIMINATION

Definitive therapy in the treatment of hyperkalemia involves potassium elimination. The routes of elimination involve the gastrointestinal (GI) tract, urine, and hemofiltration. Potassium elimination generally takes time to occur. Though it is an important step in hyperkalemia management, elimination should not be stand-alone therapy in moderate-to-severe cases as transcellular shifting therapies occur more rapidly.

GI potassium binders take advantage of the enteral-potassium reflex. Newer agents patiromer (Valtessa) and sodium zirconium cyclosilicate (Lokelma) show promise in mild-to-moderate hyperkalemia.[33] Sodium zirconium cyclosilicate's onset of action in 1 hour makes it an enticing adjunct in the emergency department management of hyperkalemia though should not be a stand-alone agent.[34] Patiromer was studied in acute hyperkalemia and its slower onset and only mild reduction make it a less ideal option than SZC.[35] Consideration of dosing of other medications should be considered, as these binders have the potential to bind other medications.[36] Older agents, sodium polystyrene sulfonate/Kayexalate, have been associated with intestinal necrosis and are not recommended given the safety of newer drugs.[37]

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In some patients, diuresis and kaliuresis can be an effective method of potassium elimination. The traditional approach is to give a loop diuretic to enhance potassium elimination, such as furosemide (Lasix). This approach is best used with patients with volume overload. Appropriate diuretic dosing is important to improve efficacy, as the final step in elimination is emergent hemodialysis. Many of the patients needing this therapy will have an element of renal insufficiency so delivering a dose that equals the degree of renal insufficiency is critical. Maximizing solute delivery at the distal nephron is crucial to the action of the diuretics. This may involve utilizing multiple agents to enhance solute delivery such as acetazolamide or a thiazide diuretic.[38]

Diuretics may still be used in those who are euvolemic in conjunction with a balanced crystalloid solution. Care should be given to avoid normal saline administration which could lead to hyperchloremic metabolic acidosis prompting worsening hyperkalemia.[39] Potassium-containing solutions (Lactated ringers and Plasma-Lyte) have been shown to be safe in hyperkalemia.[40] Isotonic bicarbonate solutions can be used here as previously described if there is a coexisting metabolic acidosis.

The final route of removal of potassium involves a form of hemodialysis. This will be the mainstay of elimination for patients who are anuric with moderate-to-severe hyperkalemia or those who fail medical management. This requires consultation with nephrology but also requires the placement of a dialysis catheter for patients without pre-existing access. These time and energy-intensive measures, though sometimes necessary, may be avoided if aggressive kaliuresis is successful.

DISPOSITION

Hyperkalemia carries a high mortality level due to the risk of life-threatening arrhythmia. Only cases of mild hyperkalemia should be considered for discharge. Although there is not a clearly evidenced based level for this, potassium 5.0 to 5.9 mmol/L without ECG changes is used in the Kidney Disease Improving Global Outcomes (KDIGO) guidelines.[13] Even among this group, patients at risk for worsening hyperkalemia due to ongoing renal failure, medication adjustment, or ongoing potassium release should still be admitted in a monitored environment. Those with ECG changes and/or severely elevated levels, over 6.5 mmol/L, should be monitored in an intermediate or ICU level environment, with strong consideration for dialysis.

SPECIAL POPULATIONS

(1) Adrenal Insufficiency

Adrenal insufficiency causes a state of hypoaldosteronism causing renal potassium retention. The classic laboratory values involve both hyponatremia and hyperkalemia. In addition to the regular treatment of hyperkalemia, giving steroids will be necessary to correct the pathology. In addition to glucocorticoid, typically hydrocortisone, administration to aid with hemodynamic response, fludrocortisone, a mineralocorticoid, should be administered as well to restore the aldosterone activity which is critical for potassium wasting. Please see Rachel E. Bridwell and Michael D. April's article, "Adrenal Emergencies," in this issue for further details.

(2) Cardiac Arrest

Patients in cardiac arrest with known or suspected hyperkalemia should be aggressively treated. The mainstay of treatment should be IV calcium as previously described. Calcium chloride should be the mainstay of therapy. The *Calcium* for Out-of-hospital Cardiac Arrest (OHCA) trial showed no benefit to calcium administration in OHCA,

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but specifically excluded patients who were suspected to have hyperkalemia, so this trial is not applicable for hyperkalemic cardiac arrest.[41] In fact, there are no randomized trials of patients in arrest with hyperkalemia, but based on case reports it is reasonable to aggressively treat with insulin and beta agonists. Additionally, patients who are refractory to this therapy may be candidates for prolonged resuscitations with mechanical cardiopulmonary resuscitation (CPR) or extracorporeal membrane oxygenation (ECMO) as a bridge to dialysis for removal of potassium. This is a very specific circumstance and involves the coordinated utilization of multiple specialties.[42]

(3) Hypothermia

Hyperkalemia may also be of prognostic value in patients with hypothermic arrest. In patients under 32° Celsius, those with a potassium over 12 mmol/L had a 100% mortality and may be used as a marker for the termination of resuscitation.[43]

(4) BRASH

The life-threatening combination of Bradycardia, Renal failure, AV nodal blockade, Shock and Hyperkalemia is termed BRASH Syndrome. Each of these issues on their own can produce derangements in potassium, but this syndrome theoretically occurs due to a viscous cycle of multiple cardiac and renal insults.[44] This should be suspected in any patient with hyperkalemia and bradycardia. Aggressive medical management to manage all deranged elements are key. In addition to standard hyperkalemia therapy, epinephrine may be the preferred agent in shock due to its action in the treatment of hyperkalemia, hypotension, and bradycardia.

HYPOKALEMIA

Defintion/burden of disease

Hypokalemia, defined as potassium levels less than 3.5 mmol/L, is often an underappreciated morbid condition. Some studies suggest that mild hypokalemia can be found in almost 15% of patients undergoing lab testing in outpatient settings, and up to 20% in hospitalized patients.[45] Frequently, the clinical relevance of this is limited as only 5% of hospitalized patients have clinically significant effects.[46] While severe hypokalemia (generally defined as <2.5 mmol/L) can have potentially deadly affects, even mild hypokalemia can lead to long term adverse outcomes in both chronic kidney disease patients and cardiac patients.[47,48] Although rare, over-exuberance in correcting a low potassium level can be dangerous, so blindly correcting this lab error should be avoided without some consideration.

Symptoms

The most common and thought about the presentation of hypokalemia is that of neuromuscular weakness. This most often presents as a complaint of “generalized weakness” which can sometimes be profound on physical exam. Muscle pain, obstipation, cramps, and palpitations/arrhythmia can all be seen as well.[49] Frank motor weakness, spasms, and fasciculations can all be present. Complaints of paresthesia or numbness, often in a pattern not consistent with a specific dermatome or myotome, can be seen as well.

Cardiac manifestations

Cardiologic complaints are common, and one of the biggest high mortality risks for hypokalemia. In fact, patients with cardiac disease, specifically heart failure, may be sensitive to low potassium levels even below 4.0 mmol/L as demonstrated by several studies.[50,51] Low serum potassium levels can lead to a variety of dysrhythmias

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including atrial fibrillation/flutter, prolonged QT interval/torsade de pointes, supraventricular tachycardia, PVCs, and ventricular arrhythmias including ventricular fibrillation. The cause of this is ultimately decreased cardiac repolarization reserve and increased intracellular calcium.[11] Typical ECG findings (**Box 1**) of hypokalemia often start with T wave flattening, followed by ST segment depression, then possible T wave inversion.[52] A prolonged QTc is common and the characteristic "U-wave," a positive deflection after a T wave, can be seen best in precordial leads. In more severe cases of hypokalemia, "Nikelekam waves" can sometimes be found on the ECG (see **Fig. 3**). These are described as an "inverse Wellen's wave, an inverted T-wave followed by a U-wave."[53] Other ECG changes that can be seen include tall P waves, prolonged QTc, and ST segment changes (particularly depression). As noted previously, sever hypokalemia can also predispose to life threatening arrhythmias including torsade de pointe, ventricular fibrillation, and ventricular tachycardia.

ETIOLOGY

Causes of hypokalemia are important to consider while taking a history and exploring possible diagnoses. Hypokalemia can be broken down into four general categories: intake disorders, GI/extrarenal losses, transcellular shifts, and renal losses. Historical components can frequently lead to suggestions of the cause.[54]

For instance, a patient that has a history of poor oral intake due to anorexia, would suggest that the hypokalemia may be caused solely by poor intake.

Malabsorptive disorders can also be considered in this category as well. Potassium is absorbed in the duodenum and jejunum, so any disease affecting the ability of these structures to function can lead to poor intake. Patients with prior diseases or bariatric surgery may have shortened or bypassed small bowel. Furthermore, decreased digestive enzymes due to bypass may affect potassium absorption.[55] These patients may need alternate supplementation methods, described further in the treatment section. Overall, hypokalemia due to poor nutritional intake is rare but can be due to severely restricted diets or eating disorders, or possibly increased need, such as during muscle regeneration from injury.[56]

Gastrointestinal loss is a slightly more common cause of hypokalemia. Patients with severe gastroenteritis may present with several days of vomiting and diarrhea, which can lead to profound hypokalemia due to poor intake as well as significant GI losses. Both vomiting and diarrheal illness can lead to low potassium levels, both through direct losses and through cellular shifting. Patients with high GI output diseases can be at risk for hypokalemia as well. This should be considered in patients with infectious diarrheal illnesses, severe gastroenteritis, pancreatitis, gastritis, and so forth.

Box 1 **ECG change in hypokalemia (Chua 2018)**
T wave flattening
Prolonged QT interval
U wave
ST segment depression
Nikelekam T waves
Torsades de pointe
Ventricular tachycardia

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Renal losses of potassium can occur due to a variety of causes. Medication induced hypokalemia is a commonly encountered ailment. Many diuretics, including all loop diuretics and thiazide diuretics, can lead to hypokalemia due to direct losses in the urinary system due to the obstructed reabsorption of potassium in the nephron. Up to 80% of patients taking diuretics will develop some level of hypokalemia.[57] Patients with hypomagnesemia and metabolic acidosis may lose potassium in their urine due to electrolyte shifting. Other endocrinologic disorders resulting in mineralocorticoid excess (increased aldosterone) result in sodium retention and potassium secretion. Renal tubule acidosis, from a variety of causes, particularly types 1 and 2 will result in urinary potassium loss as well. There are several genetic syndromes that can affect renal potassium excretion and lead to hypokalemia, outlined in **Table 4**.

Transcellular shifts can be due to several mechanisms and do not generally change the total body potassium, although the measured serum potassium will be lower. Alkalosis, respiratory or metabolic, results in low serum potassium levels. This occurs due to potassium-hydrogen exchange transporters responding to alkalosis. Insulin excess, often iatrogenic, can also lead to the shifting of potassium intracellularly without reducing total body potassium. Catecholamine excess, either due to medication use or endocrine disorder can further lead to the shifting of potassium. Rare disorders such as hypokalemic periodic paralysis can lead to transcellular shifts and are particularly susceptible to rebound hyperkalemia if overcorrected.[58]

EVALUATION

Once hypokalemia is identified, further workup in the emergency setting is rarely indicated, although it may need to be pursued in the inpatient setting if still unexplained and refractory. Much of the etiology of potassium loss can be gathered from the patient's history in order to identify the cause (above). A thorough medication history can lead to the identification of causes of intracellular shifting and increased excretion either directly or due to induced renal tubular acidosis. The history of acute gastrointestinal illness or volume losses can also provide a clear idea of the cause. Further workup, typically inpatient or outpatient includes the measurement of aldosterone-renin ratios (after potassium replenishment) can point to primary aldosteronism in patients with appropriate risk factors. In the case of persistent and refractory hypokalemia without a clear cause, workup may include the measurement of urinary electrolytes to help determine if potassium loss is renal versus nonrenal.[54]

Consider checking other electrolytes, particularly magnesium, as hypomagnesemia is extremely common in patients with hypokalemia.[57] Furthermore, specific patients with hypokalemia due to poor dietary intake are at risk for multiple electrolyte abnormalities.

Table 4
Genetic disorders leading to hypokalemia (Lin 2010)

Potassium Shifting Disorders	High Urinary Excretion
Familial hypokalemia periodic paralysis	Primary hyperaldosteronism
Andersen-Tawil syndrome	Cushing syndrome
	Congenital adrenal hyperplasia
	Liddle syndrome
	Bartter syndrome
	Gitleman syndrome

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MANAGEMENT

Treatment of hypokalemia involves replenishing potassium in order to restore homeostasis. This can be accomplished through multiple methods including IV and oral options, depending on the cause and current patient characteristics. In general, the oral method is preferred, provided the patient can tolerate oral ingestion, will absorb through the GI tract, or has access to the GI tract (nasogastric/orogastric/gastrostomy-tube) due to the high quantities of potassium that can be delivered over a period of time. Although IV supplementation has frequently been used as the treatment preference (particularly in the critical care setting), studies indicate that IV and oral management have equal efficacy in most patients.[59] IV administration is easy and sometimes necessary (**Table 5**), but not efficient. Due to direct risk of cardiovascular dysrhythmia with rapid administration, only a max of about 20 mEq/hr of potassium can be administered IV. Furthermore, especially in the case of a peripheral IV-line, potassium supplementation, typically potassium chloride, is uncomfortable for the patient as it can burn upon entry. At faster rates, there is a risk of venous sclerosis, further limiting the rate of administration.

Several formulations are available for oral supplementation, potassium chloride being the preferred and most common. This is available in pill, powder, and liquid form. In most patients, the standard pill form is adequate and will be absorbed effectively and rapidly. In patients with prior bariatric surgery, particularly Roux-en-Y bypass patients, the liquid or powder formulation may be preferred due to more rapid proximal absorption compared to pills.[55] Liquid formulations often are associated with unpleasant taste, to the extent that it may not be tolerated by some patients. Potassium phosphate supplementation is available as well but is less effective in replenishing hypokalemia associated with chloride loss, which is most likely in many patients. Potassium bicarbonate formulations are also available and may be preferred in the setting of metabolic acidosis.[60] Significant risks of potassium supplementation are relatively rare. Hyperkalemia due to overcorrection can certainly be a concern, especially in patients with poor renal function or hypokalemia due to intracellular shifts. Gastrointestinal discomfort is the most likely side effect, usually improved if taken with food/water. In severe instances, ulceration can occur.

The amount of potassium needed to treat depends on the degree of hypokalemia present. For instance, in mild hypokalemia (3.0–3.4 mEq), the total body deficit may only be 100 to 200 mEq. However, lower levels of potassium can be reflective of significant total body potassium deficit, possibly up to four to five times as much, depending on the level.[61] Furthermore, an easy mistake is to discount concurrent electrolyte abnormalities in the correction. Patients that are significantly hypokalemic are

Table 5
Potassium replenishment options

Drug	Route	Benefit
Potassium Chloride	Oral	Most common, useful with metabolic alkalosis
Potassium Chloride	Intravenous	If no gut access, severe levels needing emergent treatment, profound shock. Cardiac arrest or unstable arrhythmia
Potassium Bicarbonate/ Citrate	Oral	Equal efficacy to replenish potassium. Can improve metabolic acidosis
Potassium acetate	Intravenous	Bicarbonate precursor, so same as above
Potassium phosphate	Intravenous/ Oral	Concurrent hypokalemia and hypophosphatemia

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almost certainly significantly hypomagnesemic as well and will not be able to retain the potassium supplementation until their hypomagnesemia is corrected. Concurrent administration of magnesium is often needed in severely hypokalemic patients. Several different formulations of potassium supplementation are available (see Table 5). Overall, potassium chloride is the preferred formulation due to the additional chloride that can aid in treating concomitant metabolic alkalosis.

Disposition

In patients with hypokalemia, the need for admission is based around several factors. If the identified cause is treated and managed, with appropriate ability to maintain or safely supplement potassium at home, then many patients with mild-to-moderate hypokalemia can often be safely discharged. Patients with increased risk for arrhythmia due to severe hypokalemia (<2.5 mmol/L), ECG changes, arrhythmia-inducing drugs (See Digoxin later in discussion), or inability to cease potassium loss or adequately supplement the potassium should be considered for admission and telemetry monitoring. Severe persistent hypokalemia, presence of arrhythmia, or condition that would lead to the persistent critical decrease of potassium should be considered for intermediate or ICU level monitoring.

SPECIAL POPULATIONS

(1) Diabetic Ketoacidosis

A patient with diabetic ketoacidosis (DKA) is already critically ill. A DKA patient with hypokalemia is even more problematic. DKA treatment requires insulin administration, often as a continuous infusion, or frequent redosing (See Bobbi-Jo Lowie and Michael Bond's article, "Diabetic Ketoacidosis," in this issue for further details). Administration of insulin, as discussed previously, results in effective intracellular shifting of potassium. If the patient's potassium level is already low, this can lead to life threatening arrhythmia and cardiovascular collapse. It is recommended to hold insulin administration until the potassium level is known. Insulin should not be started (or should be held) if a level less than 3.3 mmol/L is identified, until potassium level is safely increased to start insulin therapy. Even the potassium level under 5.3 mmol/L, despite being "normal range," should prompt concurrent IV administration of potassium chloride with IV fluids.[62]

(2) Renal failure

In patients with either acute or chronic kidney disease, particularly severe renal failure, hypokalemia can still be present, although they are at much greater risk for hyperkalemia. Because of this risk, it is overall unlikely that the patient's potassium level would continue to fall and would in most cases not fall rapidly. Goals of potassium replenishment are still similar, with a goal greater than 3.5 mmol/L, as lower levels can lead to further ESRD, however care must be taken to not overcorrect.[63]

(3) Cardiac Arrest

Patients in cardiac arrest due to hypokalemia-induced arrhythmias will need rapid IV potassium replacement. Recommended rates are much faster than typically noted. Potassium chloride IV 20 mEq over 2 to 3 minutes for adults, repeating until $K > 4.0$ mmol/L has been suggested.[24] European resuscitation guidelines for pediatrics recommend 1 mmol/kg (max 30 mmol) over 20 minutes in the peri-arrest state until a goal of 2.5 mmol/L. They do not provide specific recommendations for faster

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administration during an arrest with known severe hypokalemia[64] Concurrent rapid administration of IV magnesium is recommended for both adult and pediatric populations.

(4) Digoxin

Patients on digoxin therapy are at significantly increased risk for any potassium-induced arrhythmias.[65] Potassium goals for these patients should be aggressively maintained above 3.5 mmol/L-6.0 mmol/L.[66] Digoxin may need to be held in severely hypokalemic patients as even at therapeutic levels of digoxin, concurrent hypokalemia can lead to a dangerous arrhythmia.

SUMMARY

Patients with potassium irregularities are commonly encountered in the ED. Potassium levels are closely regulated by aldosterone and alterations in renal physiology are linked to serum changes. At their extremes, potassium disorders both hypokalemia and hyperkalemia can cause deadly cardiac arrythmias due to their disruption of the resting membrane potential. An ECG and ultimately serum lab values will guide emergency treatment while searching for an inciting cause.

CLINICS CARE POINTS

- ECG is likely the fastest test to signal a potassium derangement and need for emergent treatment.
- Hyperkalemia can be rapidly fatal and requires prompt treatment and evaluation of its underlying cause. Membrane stabilization, transcellular shifting, and elimination are the cornerstones of hyperkalemia treatment.
- Do not forget to replenish magnesium along with potassium replenishment
- It is easy to underestimate the total body potassium deficit. As serum potassium levels drop, the total body defect is decreased in a logarithmic manner, not linear. More potassium supplementation is needed relative to mild hypokalemia.

DISCLOSURE

Neither author has any financial relationship to disclose.

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